Available online on 15.04.2024 at http://jddtonline.info

Journal of Drug Delivery and Therapeutics

Open Access to Pharmaceutical and Medical Research

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Review Article

Understanding Vitiligo: Causes, Diagnosis, Promising Advances in Treatment and Management and the Impact on Mental Health

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Article Info:

Article History:

Received 19 January 2024
Reviewed 04 March 2024
Accepted 22 March 2024
Published 15 April 2024

Cite this article as:

Rama Rao T, Sharmeen H, Tabassum A, Jasmine SJ, Understanding Vitiligo: Causes, Diagnosis, Promising Advances in Treatment and Management and the Impact on Mental Health, Journal of Drug Delivery and Therapeutics. 2024; 14(4):130-137

DOI: http://dx.doi.org/10.22270/jddt.v14i4.6503

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Abstract

Vitiligo is a chronic skin disorder characterized by the loss of pigmentation, resulting in white patches on the skin. The condition arises from the destruction of melanocytes, the cells responsible for producing melanin, the pigment that gives colour to the skin, hair, and eyes. Although the exact etiology of vitiligo remains elusive, it is believed to involve a complex interplay of genetic, autoimmune, and environmental factors.

This overview provides the key aspects of vitiligo, including its epidemiology, pathogenesis, clinical manifestations, and current treatment approaches. The prevalence of vitiligo varies globally, affecting individuals of all ethnic backgrounds and age groups.

The autoimmune hypothesis proposes that the immune system mistakenly targets and attacks melanocytes, leading to their destruction. Triggering factors, such as oxidative stress, viral infections, and neural factors, may contribute to the initiation and progression of vitiligo.

Clinically, vitiligo can significantly impact the quality of life for affected individuals, both psychologically and socially. The distribution and extent of involvement vary, and the condition can affect any part of the body.

Various treatment modalities are available for vitiligo, ranging from topical corticosteroids and calcineurin inhibitors to phototherapy and surgical interventions. While these approaches may help to re-pigment the skin to some extent, there is no definitive cure, and the outcomes can be unpredictable. Ongoing research is focused on understanding the molecular mechanisms underlying vitiligo and developing targeted therapies to address the root causes of the disease.

Keywords: Vitiligo, Autoimmune, Melanocytes, Pigmentation, Corticosteroids, Quality of life.

INTRODUCTION

Derived from the Latin word "vitium," signifying a "blemishing fault," vitiligo manifests as white patches, impacting self-esteem and perpetuating stigma. This chronic pigmentary disorder primarily affects melanocytes in the skin and mucosae, with rare involvement in the eyes and inner ear labyrinth. Its origin lies in the intricate interplay of genetic, environmental, and autoimmune factors [1].

Vitiligo is a persistent skin condition characterized by epidermal pigmentation loss, perceived as disfiguring and burdensome by those affected. Despite the psychological strain and treatment needs, population-based epidemiology has given little attention to the disease. Epidemiological data often focuses on selected environments or comorbidity prevalence without considering the general public. Studies suggest vitiligo associations with atopic dermatitis, alopecia areata, and psoriasis [2].

While there is no cure for vitiligo, various management strategies aim to reduce white patch spread and re-pigment affected areas. Cost-effective strategies are still needed to prevent vitiligo resurgence and halt white patch spread. Understanding molecular mechanisms and patients' environmental context is crucial for effective treatment development.

Melanocytes in vitiligo patients experience oxidative stress due to excess reactive oxygen species from defective mitochondria and a deficient endogenous antioxidant system. This redox imbalance causes substantial damage to proteins, lipid membranes, and DNA. Damaged melanocytes release damage-associated molecular patterns, triggering an inflammatory gene expression response that ultimately leads to cell apoptosis. [3].

EPIDEMIOLOGY

Global prevalence figures for vitiligo exhibit a broad range, spanning from 0.004% to 2.28% [4]. This condition affects both genders equally, typically emerging before the age of twenty in approximately half of cases [1]. Vitiligo is widespread across ethnicities and skin types, yet geographic variations are notable. For instance, prevalence in China's Shaanxi Province is reported at 0.093%, while certain areas in India show rates as high as 8.8% [5]. The prevalence is notably higher in young women, peaking in early adolescence, while males reach their peak between 45 and 60 [6].

CLASSIFICATION AND TYPES

As per the Vitiligo Global Issues Consensus Conference held between 2011-2012, vitiligo canbe categorized into two main types: [7]

1. **Segmental Vitiligo:**

Segmental vitiligo lesions exhibit a unilateral, segmental, or band-shaped distribution. It includes:

a. **Focal Vitiligo:** Involves the discoloration of one or more parts on a single body.

b. **Mucosal Vitiligo:** Primarily affects mucous membranes.

2. **Non-Segmental Vitiligo:**

Lesions are bilaterally distributed in an acrofacial pattern or scattered symmetrically across the entire body. This type is further divided into six categories:

a. **Acrofacial:** Mainly affects the face, head, feet, and hands, particularly involving the perioral region.

b. **Mucosal:** Affects oral and genital mucosa, with areas of mucosa potentially impacted in acrofacial cases.

c. **Generalized or Common:** Can affect any skin part, predominantly hands, fingers, face, and trauma-exposed areas.

d. **Universal:** Impacts about 80-90% of the entire body and is the most common form in adulthood.

e. **Mixed:** Displays both segmental and non-segmental vitiligo, with the segmental form often preceding NSV.

f. **Rare Forms:** Encompass punctata, minor, and follicular types, considered unclassifiable.

Figure 1: Types of vitiligo

AETIOLOGY AND PATHOGENESIS

Vitiligo exhibits a complex origin and unpredictable progression, responding to various endocrine signals. Although multiple genes contribute to this condition, their triggers can vary. Psychological stress, a notable factor, can generate reactive oxygen species through both external and internal stimuli.

Melanocytes, the specialized pigment-producing cells in the skin, generate melanosomes containing melanin pigments. The compartmentalization of melanosomes serves to protect cells from byproducts like quinines and hydrogen peroxide. Mature melanosomes are transferred to keratinocytes, and melanocytes' interactions with keratinocytes and dermal fibroblasts influence their properties. Factors like stem cell factor and neuregulin 1 regulate melanocytes, while interleukin-1 (IL-1), interleukin-6 (IL-6), and tumour necrosis factor- alpha (TNF-α) inhibit melanogenesis. α-melanocyte-stimulating hormone and eicosanoids enhance melanin synthesis.

Melanocytes are vulnerable to oxidative stress, viral infections, and undergo continuous replacement. Immune-mediated destruction of melanocytes has been observed following melanoma. Among various autoimmune pathologies, thyroid diseases, including dermatitis herpetiformis, urticaria, alopecia areata, and vitiligo, affect the skin. Generalized vitiligo is linked to autoimmune thyroid diseases such as Hashimoto's and Graves' disease [8].

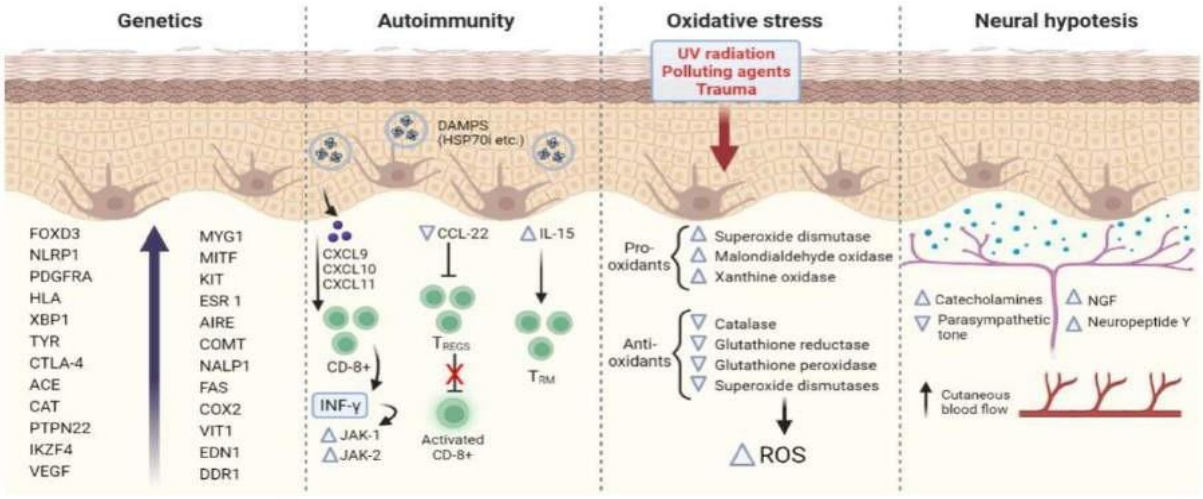

Figure 2: Vitiligo pathogenesis

1) AUTOIMMUNE Innate Immunity

Genome-wide association studies conducted on individuals with vitiligo have identified multiple susceptibility loci associated with genes that regulate innate immunity. Recent studies have demonstrated the significance of HSP70i in vitiligo pathogenesis in a mouse model, highlighting its role in inducing inflammatory dendritic cells (DCs).

These DCs may, in turn, exhibit cytotoxicity or carry and present melanocyte-specific antigens to T cells in lymphoid tissues. This interaction is considered a crucial crosstalk step between innate and adaptive immunity, ultimately leading to T cell-mediated autoimmune destruction of melanocytes.

Adaptive Immunity

Cytokines released within the skin serve as an early signal, aiding these autoreactive T cells in locating stressed melanocytes. IFN-γ and IFN-γ-induced chemokines (CXCL9 and CXCL10) exhibit high expression in both the skin and blood of vitiligo patients and a mouse model. Furthermore, IFN-γ and CXCL10 are essential for both disease progression and maintenance in the mouse model.

A recent study found that serum CXCL10 levels were not only elevated in vitiligo patients compared to healthy controls but were also associated with disease activity [9].

1) GENETIC THEORY

Familial clustering is evident in vitiligo, with the prevalence among first-degree relatives ranging from 0.14% to 20%, indicating a clear genetic component. However, the fact that only 23% of monozygotic twins exhibit concordance suggests a significant non-genetic factor in vitiligo's pathophysiology. Given its polygenic nature, various candidate genes have been identified, including the major histocompatibility complex, angiotensin-converting enzyme, human leukocyte antigen, catalase (CAT), cytotoxic T lymphocyte antigen-4 (CTLA-4), protein tyrosine phosphatase, and interleukin-2 receptor A (IL2RA). All these genes, involved in immune control, have been examined for their genetic association with generalized vitiligo.

2) OXIDATIVE STRESS THEORY

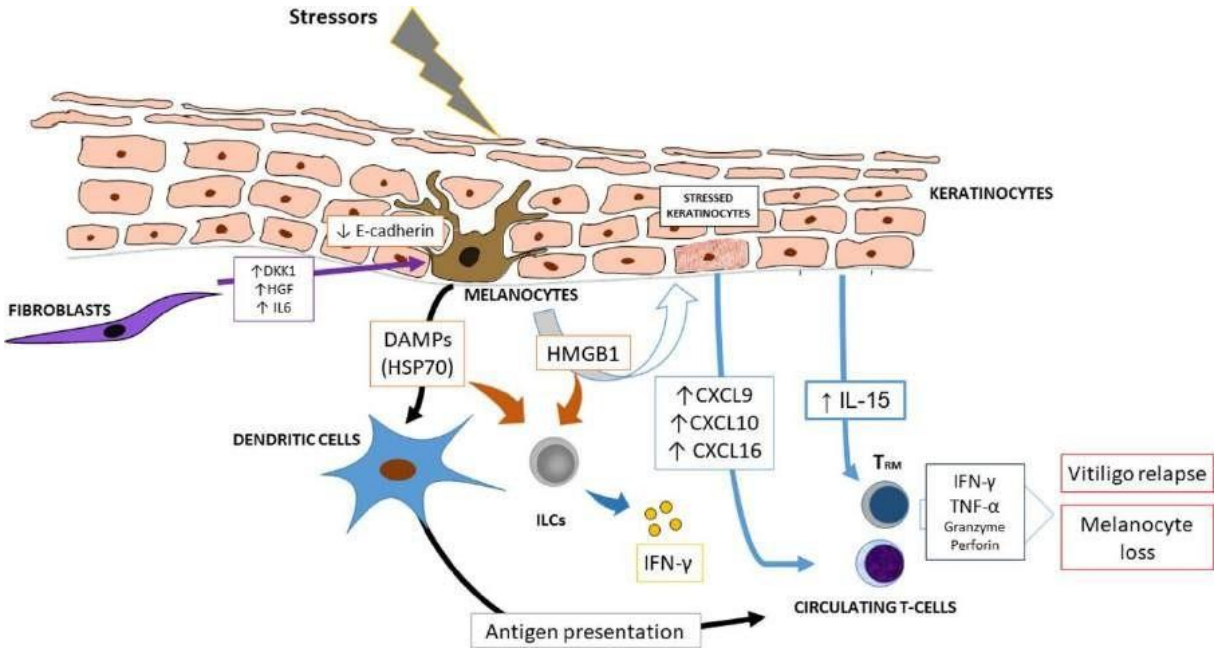

Figure 3: Oxidative stress theory

As per the oxidative stress theory, intra-epidermal build-up of reactive oxygen species, particularly hydrogen peroxide, at concentrations as high as one millimole, is the main pathophysiological component of vitiligo. Malondialdehyde (MDA), selenium, vitamins C and E, glutathione peroxidase, superoxide dismutase (SOD), and catalase (CAT) are important indicators of interest. Lipid peroxidation produces MDA, which is a byproduct that indicates oxidative stress. Superoxide radicals are neutralized by SOD, which lessens their toxicity, and they are converted into oxygen and water by CAT. Patients with vitiligo have markedly increased levels of SOD, decreased erythrocyte GPx activity, decreased CAT levels, and decreased serum and epidermal levels of vitamins C and E.

3) NEURAL THEORY

The neural theory suggests that neurochemical substances released by nerve endings can either decrease melanin production or harm melanocytes. Additionally, it posits a connection between the pathogenesis of vitiligo and the catalase gene. Catalase, an enzyme found in nearly all living organisms, plays a role in hydrolysing hydrogen peroxide into water and oxygen, providing protection against highly reactive oxygen radicals. In both lesional and non-lesional skin of vitiligo patients, there is a decrease in catalase enzyme activity.

4) BIOCHEMICAL THEORY

The biochemical theory proposes that the accumulation of toxic intermediate metabolites during melanin synthesis and insufficient defence against free radicals result in elevated levels of hydrogen peroxide, leading to the destruction of melanocytes.

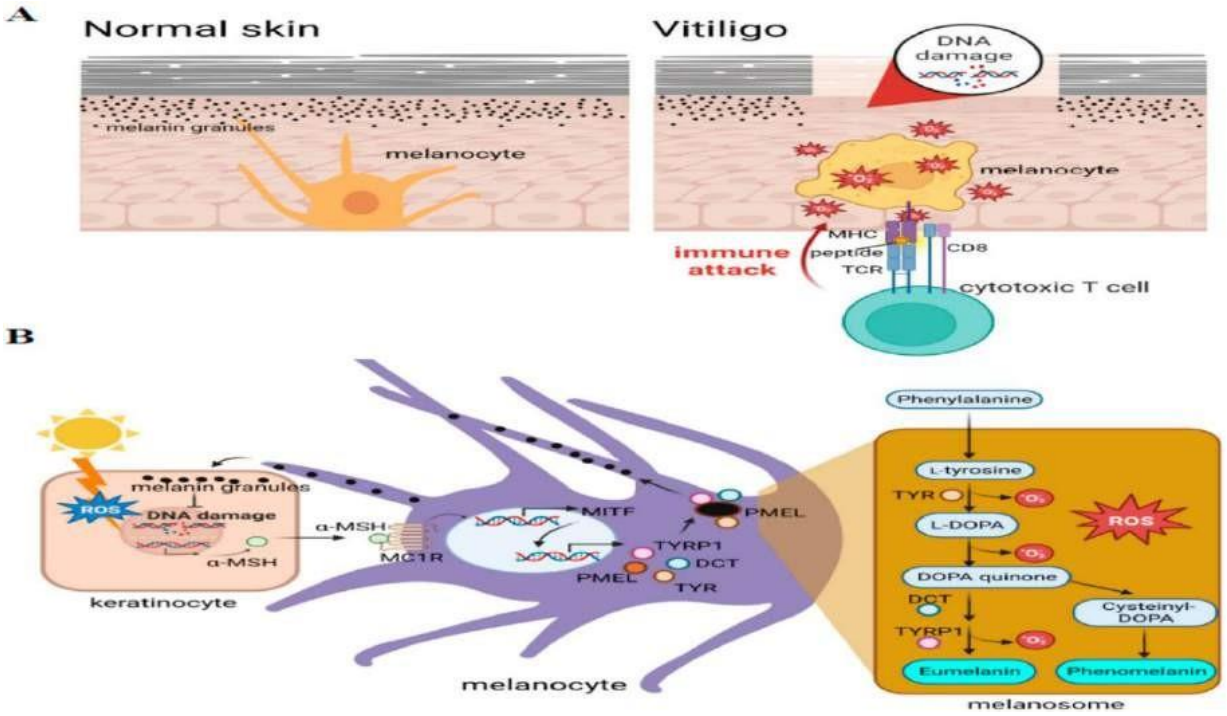

Figure 4: The role of oxidative stress in the development of vitiligo

Some theories suggest a combination of genetic factors, flaws in melanocyte structure and function, and insufficient melanocyte development factors contribute to the depigmentation process. However, the manifestation of the disease is influenced by environmental factors [10].

SIGNS AND SYMPTOMS

The primary indication of vitiligo is the presence of one or more areas of lighter skin, which is often the sole manifestation for many individuals. Additional signs and symptoms may emerge, including: [11]

- Development of spots and patches of lighter skin
- Transformation of patches into a white hue
- Presence of lighter patches inside the mouth or nose
- Increased susceptibility of patches and spots to sunburn
- Itching associated with the patches
- Greying or whitening of hair
- Onset of hearing loss
- Alterations in eye colour

DIAGNOSIS HISTOPATHOLOGY

The basic histological findings of vitiligo are the absolute loss of epidermal pigmentation due to the complete lack of functioning melanocytes. For further study, melanocyte-specific electron microscopy and immunohistochemistry with markers like Melan-A and HMB-45 can be used. Melanocytes exhibit cytoplasmic vacuolization, elongated dendritic processes packed with melanin granules, and cellular expansion as degenerative alterations. Further documented changes include epidermal vacuolization, an increase in Langerhans cells, and basement membrane thickening.

I. PHYSICAL EXAMINATION

Vitiligo is typically diagnosed through clinical examination based on the following characteristics: [12]

- Macules or patches exhibit a convex border and lack signs of inflammation, with adjacent normal skin.
- Milky white-coloured macules are ranging from few millimetres to several centimetres, commonly on the face, neck, dorsum of hands, scalp, and trunk.
- Lesions can also manifest in trauma-prone areas such as knees and elbows.
- Koebnerization may occur in 20% to 60% of vitiligo patients.

II. MEDICAL AND FAMILY HISTORY

- History of symptoms: history of disease onset, its growth is inquired. In some cases, premature greying of hair is observed.
- Vitiligo in the family: 20% of affected individual are close family member with the condition.
- Family history of autoimmune diseases: inquire about a family history of autoimmune diseases such as rheumatoid arthritis (RA), lupus, or thyroid disease.
- Past skin problems: This includes instances of severe sunburn, rashes, or other traumas in areas of white or light patches.
- Stress levels: Physically, mentally, and emotionally stressful situations, may also contribute.[13].

III. DIAGNOSTIC TESTS AND PROCEDURE

To correctly diagnose vitiligo, a number of diagnostic procedures and tests are carried out. Like the Wood's light test, which exposes skin depigmentation by employing a portable UV lamp that releases UVA. Because it can identify areas of pigment loss that are invisible to the unaided eye, this test is especially helpful for pale skin tones [14].

A skin biopsy is an additional diagnostic procedure that involves taking a tiny sample of the afflicted skin tissue in order to look for melanocytes, which are pigment cells. The

presence or absence of these cells is then assessed under a microscope in the laboratory on the skin sample.

In some cases, blood tests may be requested, including a complete blood count and an antinuclear antibody test to assess overall health and address specific concerns. Finally, if an individual experiences symptoms related to vision or hearing, assessment of potential eye inflammation (uveitis) or an audiologist for hearing tests is done. [13].

IV. DIFFERENTIAL DIAGNOSIS OF VITILIGO

Vitiligo is not the only condition that can cause skin depigmentation. Therefore,dermatologists explore alternative conditions if clinical signs or test outcomes deviate from a typical vitiligo case. Other pigment disorders that may cause skin depigmentation include Nevus depigmentosus, a segmental depigmented or hypopigmented region present at birth; Pityriasis alba, which mainly affects children and manifests as white, scaly macules on the face and sun-exposed areas; Tinea (pityriasis) Versicolor, a superficial fungal infection that causes pigment loss and presents as pale, scaly macules on the back and chest that fluoresce yellow under Wood's lamp; Halo nevus, a melanocytic nevus surrounded by an oval halo of depigmentation; Progressive macular hypomelanosis, which presents as asymptomatic hypopigmented patches on the trunk in young adults[12].

TREATMENT

A. TOPICAL THERAPY Calcineurin inhibitors

Calcineurin inhibitors, acting as immunomodulators, serve as an off-label remedy for vitiligo. Their mechanism involves inhibiting calcineurin, a pro-inflammatory protein in lymphocytes and dendritic cells responsible for the transcription of interleukin (IL)-2 and tumour necrosis factor-α (TNF-α). By inhibiting calcineurin, cytokine production decreases, fostering melanocyte and melanoblast proliferation [15].

Corticosteroids

Corticosteroids, particularly potent and very potent topical ones, are the primary treatment for vitiligo, acting by modulating and inhibiting inflammation. Treatment response is more favourable in sun-exposed areas, while acral regions may show a poorer response. High- potency corticosteroids are recommended for small body areas, and in sensitive regions, topical calcineurin inhibitors or lower-potency steroids are preferred. The suggested application is daily for up to 3 months, followed by intermittent use for up to 6 months. A meta-analysis indicates comparable effectiveness of potent and very potent corticosteroids in achieving ≥75% repigmentation in localized vitiligo.

Pseudocatalase/superoxide dismutase

Oxidative stress and the buildup of hydrogen peroxide (H_2O_2) are thought to be contributing factors in vitiligo. This accumulation proves toxic to melanocytes, inhibits tyrosinase, and leads to the deactivation of catalase, an enzyme within peroxisomes that facilitates the reduction of H_2O_2 to water and oxygen. Hence pseudocatalase reduces H_2O_2 to water and oxygen

Methotrexate

Methotrexate acts as a folate antagonist, seemingly reducing the T cell count producing TNF- α, thereby exhibiting anti-inflammatory, immunomodulatory, and antiproliferative effects. A recent case report detailed substantial repigmentation in a stable vitiligo patient treated with topical methotrexate 1% gel twice daily for 12 weeks, along with folic acid supplementation.

Prostaglandin F2 alpha analogues

The utilization of prostaglandin F2 alpha analogues is widespread in treating ocular hypertension. The application of these analogues in vitiligo stemmed from the recognition of iris and periocular skin hyperpigmentation in glaucoma patients. This heightened pigmentation appears to result from an augmentation in melanogenesis.

B. SYSTEMIC TREATMENT Corticosteroids

The primary goal of treatment employing systemic corticosteroids (SCS) is immune responsesuppression, aiming to stabilize the disease and promote repigmentation. SCS is prescribed for the treatment of rapidly progressive active vitiligo. Utilizing pulse therapy with SCS is preferred to mitigate potential side effects.

Statins

Statins, known for their lipid-lowering properties, contribute to the treatment of vitiligo by exerting anti-inflammatory and immunomodulatory effects. This involves inhibiting CD8 T-cell proliferation, chemokines, proinflammatory mediators, and the expression of proinflammatory adhesion molecules. Additionally, they reduce IFN-γ production, leading to decreased expression of major histocompatibility complex II and inhibition of activated T cells [16].

C. LASER THERAPY

The excimer laser utilizes monochromatic rays at 308 nm to address limited, stable patches of vitiligo. This innovative treatment proves effective, safe, and well-tolerated; however, it comes with a substantial cost. For localized vitiligo lesions, the therapy involves biweekly sessions, typically totalling 24-48 sessions. In cases of segmental vitiligo, known for being more resistant to repigmentation in some patients, the excimer laser has been combined with both topical tacrolimus and short-term systemic corticosteroids. Studies indicate that utilizing the excimer laser treatment at earlier stages of the disease enhances the repigmentation response in segmental vitiligo [15, 17].

D. PHOTOTHERAPY

UVB irradiation, falling within the wavelength range of 280–320 nm, holds more significance than UVA (with a wavelength of 320–400 nm). In contemporary practice, narrow-band UVB is frequently employed in conjunction with topical corticosteroids (TCS) or calcineurin inhibitors (TCI) for treating various subtypes of vitiligo. Phototherapy options for vitiligo encompass PUVA and UVB (narrow-band UVB (NB-UVB), excimer laser, or lamp). While PUVA was the initial successful phototherapy protocol, it carries drawbacks such as inapplicability to children or pregnant women and phototoxic side effects like nausea, headache, dizziness, and an increased risk of skin cancer. In comparison, NB-UVB has demonstrated higher treatment efficacy and is associated with fewer and milder side effects [10, 18].

E. SURGERY

Significant progress has been made in vitiligo surgical treatments, with tissue grafts involving direct transfer from pigmented areas and cell transplants requiring processing. Various tools and techniques are employed, with donor sites often discreet. Dermatosurgery is effective for segmental vitiligo, and clinical results are enhanced with NB-UVB or PUVA phototherapy post-surgery. Guidelines by the British Association of Dermatologists in 2021 recognize surgery as an option, particularly for stable segmental or non-segmental vitiligo when other methods are inadequate. However, surgical therapies are weakly recommended interventions due to limited availability and specialization.

Multiple surgical approaches are available for treating vitiligo, aiming for comprehensive repigmentation to aesthetically match the surrounding unaffected skin. The selection of a surgical method is contingent upon factors such as the extent of vitiligo, the affected areas. Below, are different surgical techniques, encompassing tissue grafts and cellular grafts [19].

Grafting Techniques

- Tissue grafting methods include Mini punch grafting, Suction blister grafting, thin split-thickness grafting, Hair follicle grafts, Mesh grafts, and Flip-top pigment [20].
- Cellular grafting options consist of non-cultured basal cell suspensions and Cultured melanocytes/keratinocyte grafts [19].

F. DEPIGMENTATION THERAPIES

These treatments are typically recommended for extensive and persistent vitiligo, especially when more than 50% of the body surface is affected or when cosmetically sensitive areas are prominently involved.

Initially, Monobenzyl ether of hydroquinone (MBEH) 10% concentration is applied topically daily for the first month, followed by daily application of MBEH at 20% for another month. If the areas show limited response and are well-tolerated, the concentration can be increased to 30-40%. Generally, patients experience depigmentation in areas distal to the application after 3-6 months. Alternative treatment options include 4-methoxyphenol, 88% phenol solution, laser, and cryotherapy [16, 21].

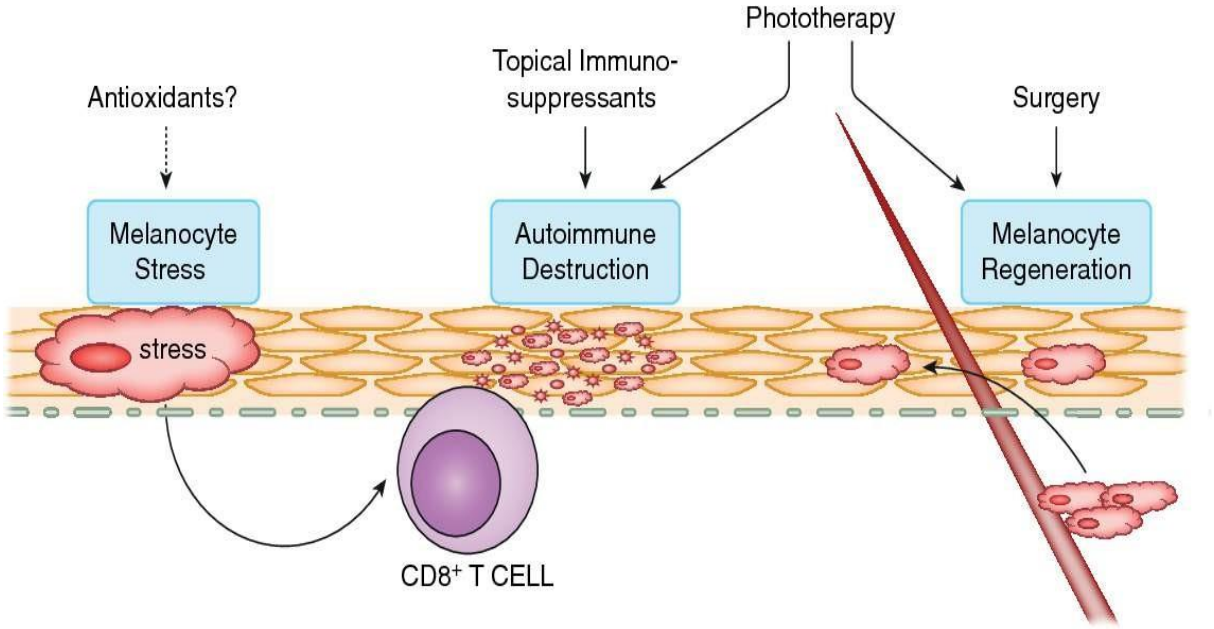

Figure 5: Treatment considerations for vitiligo

EMERGING TREATMENTS

In light of our present comprehension of vitiligo's pathogenesis, an effective approach to treating the condition should encompass three key strategies: alleviating stress on melanocytes, modulating the autoimmune response, and promoting the regeneration of melanocytes.

While current treatments partially meet these requirements, upcoming therapies have the potential to address them more precisely, and combining therapies might synergistically enhance the overall treatment response.

Regulating Autoimmunity

The advancement of immunomodulators for addressing inflammatory skin conditions involves the exploration of more focused treatments. Recent progress in comprehending the immunopathogenesis of vitiligo has enabled the identification of novel immune targets, paving the way for the development and testing of innovative treatments for vitiligo.

HSP70i

A particular study highlighted the involvement of the heat shock protein HSP70i in the development of vitiligo, proposing that stressed melanocytes release it, triggering innate inflammation in the skin.

Subsequent experiments showed that altering the protein reduced its immunogenicity and seemed to foster tolerance when expressed in the skin of a mouse model, preventing disease onset. The researchers suggested potential future exploration of this approach as a novel treatment for vitiligo, though the delivery of a mutant protein via DNA in patient skin may require time for development and safety demonstration.

JAK-STAT Signalling

The JAK-STAT signalling pathway is crucial for relaying extracellular signals from various cytokines, such as IFN-γ, to the nucleus. Upon cytokine receptor binding, Janus kinases (JAKs) phosphorylate signal transducer and activator of transcription proteins (STATs), activating them to initiate the transcription of target genes.

The JAK family comprises four members: JAK1, JAK2, JAK3, and Tyrosine kinase 2 (TYK2). Specifically, JAK1 and JAK2 play direct roles in IFN-γ signalling, activating STAT1 and leading to the transcription of IFN-γ-induced genes, including CXCL10.

WNT Signalling

In a recent investigation, it was observed that melanocytes in individuals with vitiligo exhibited compromised WNT signalling—a pathway crucial for fostering the differentiation of melanocyte precursors in the skin.

The researchers theorized that this impaired signalling played a role in the disease's pathogenesis, specifically hindering melanocyte regeneration and repigmentation during treatment. Experiments utilizing explanted human skin ex vivo indicated that activating WNT could enhance melanocyte differentiation. Consequently, therapeutic WNT activation might serve as a supplementary approach for vitiligo treatment, supporting melanocyte regeneration [9].

MANAGEMENT AND COPING STRATEGIES

CAMOFLAGUE TECHNIQUES

Camouflage originates from the French term 'Camoufler', signifying 'to blind' or 'veil'. Referred to as protective concealment, it involves masking an object in plain sight to obscure it from observation. Furthermore, the application of camouflage enhances an individual's visual presentation, shifting the focus away from undesirable discoloration, and consequently boosting confidence and self-esteem.

Temporary camouflage options encompass liquid dyes, native formulations, remedial cosmetic camouflage, and self-tanning products. Acting as a swift and non-invasive intervention, skin camouflage effectively mitigates the emotional distress of patients by concealing vitiligo patches [22].

SELF CARE MEASURES

Vitiligo's unpredictability makes it crucial to focus on controllable behaviours for improved well-being. Consider the following guidelines: [23]

a. Safeguard skin from the sun, as its prone to sunburn by seeking shade, wearing protective clothing, and applying sunscreen.

b. Steer clear of cuts, scrapes, and burns, as these may trigger new vitiligo spots.

c. If desiring added colour, opt for safe options like camouflage makeup, self-tanner, or skin dye.

d. Be aware of tattoo risks, as skin injuries during the process can induce the Koebner phenomenon, leading to new vitiligo spots.

e. Support the immune system through stress reduction and a balanced diet.

f. Prioritize mental health using counselling or support groups.

g. Educate yourself about vitiligo, including treatment options and factors influencing its spread.

LIFESTYLE CONSIDERATION

Extensive research has been conducted on various alternative treatments, including herbal products and vitamin supplements, to complement traditional therapeutic approaches for vitiligo. Some of these alternative treatments include Vitamin B12 & Folic Acid [24], Vitamin C [25], Vitamin D, and Zinc.

Vitamin B12 & Folic Acid which help with DNA synthesis, repair, and methylation, and have been found to induce repigmentation when supplemented with sun exposure. Vitamin D helps with immune system function and has been found to decrease disease progression when supplemented with standard therapy.

Finally, Zinc hinders the destruction of melanocytes. While it yields slight benefits when used in conjunction with topical steroids, it is important to consult with a healthcare professional before starting any alternative treatments [26].

HERBAL PRODUCTS

Vitiligo management can be significantly impacted by the use of herbal products as supplements. Many herbal compounds have varied qualities, including anti-inflammatory, antioxidant, photoprotective, immunomodulatory, and stimulatory effects on melanogenesis. Examples of these items include Polypodium leucotomos, Khellin, Phenyl alanine, Phyllanthus emblica, Piperine, Nigella sativa, and Curcumin. When combined with UV therapy, khellin activates melanocytes and helps to reduce depigmented lesions. Conversely, piperine causes melanocytes to proliferate and starts the process of dendritic development in melanocytic cells. However, these natural remedies can make a big difference in how Vitiligo affects you [6].

POTENTIAL COMPLICATIONS

Patients with vitiligo exhibit a higher prevalence of autoimmune thyroid conditions compared to the general population. The sensitivity of anti-TPO antibodies in detecting early subclinical forms of autoimmune thyroiditis makes them valuable for identifying individuals at an elevated risk of developing such pathologies. The influence of autoimmune thyroid pathologies (and psoriasis) significantly impacts the extent of skin depigmentation in vitiligo.

Notably, autoimmune thyroid pathologies emerge as the sole coexisting autoimmune or inflammatory diseases in vitiligo patients exhibiting a distinctive distribution pattern. Recent emphasis on the Th1-type immune response and the role of C-X-C Motif Receptor 3 (CXCR3) and its corresponding chemokine, CXCL10, has underscored their significance in the pathogenesis of vitiligo [27].

IMPACT ON QUALITY OF LIFE

Vitiligo affects individuals of all races, with a heightened impact on dark-skinned people due to pronounced contrast. The visibility of chronic dermatoses in vitiligo leads to daily stigmatization, causing psychosocial stress and potential depression. The chronic, visible, and relapsing nature of vitiligo impairs both quality of life and coping abilities, with severe depression even leading to suicidal thoughts. Patients often hesitate to report psychological distress, resulting in a greater focus on physical symptoms rather than on psychological aspects like depression, stress, or stigmatization [10].

Research indicates that a substantial percentage of dermatology patients with vitiligo suffer from psychiatric comorbidity, emphasizing the need for addressing emotional effects in treatment. Treating vitiligo should incorporate psychological intervention to enhance adaptation and overall quality of life. A holistic approach is essential to improve the emotional well-being of patients, reduce the feeling of stigmatization associated with this chronic condition [28, 29].

CONCLUSION

Vitiligo remains a challenging dermatological condition with multifaceted origins. Advances in genetics, immunology, and epigenetics are shedding light on its complex pathogenesis. The development of more effective and targeted treatments holds promise for improving the lives of individuals affected by vitiligo. Further research is needed to unravel the intricate mechanisms involved in the onset and progression of this enigmatic skin disorder.

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